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ROENTGEN DIAGNOSTICS OF NECROTIZING ENTEROCOLITIS (NEC) IN NEWBORNS.

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Abstract:

This is a non-specific inflammatory disease caused by infectious agents against the background of immaturity of local defense mechanisms and hypoxic-ischemic damage to the intestinal mucosa. Mortality ranges from 27 to 35%. Necrotizing enterocolitis (NEC) of newborns (Necrotizing enterocolitis, necrotizing enterocolitis, ulcerative necrotic enterocolitis, "disease of surviving prematurity) is a nonspecific inflammatory disease caused by infectious agents against the background of immaturity of local defense mechanisms and hypoxic-ischemic damage to the intestinal mucosa, prone to generalization of the process with development of a systemic inflammatory response.

Keywords: GIT - Gastrointestinal tract;NN - Premature newborn;NEC - Necrotizing Enterocolitis;OBP - Abdominal Organs;OPN - Department of Neonatal Pathology

Necrotizing enterocolitis (NEC) of newborns is a nonspecific inflammatory disease caused by infectious agents against the background of immaturity of local defense mechanisms and hypoxic-ischemic damage to the intestinal mucosa. The mortality rate ranges from 27 to 35%. Early diagnosis is based on the clinical picture, X-ray examination of the abdominal cavity, laboratory test data. The popular classification according to M. Bell (1979) is presented in the modification of M. Walsh and R. Kliegmap (1986), which is of great importance in choosing a treatment method. Information is given on the etiology, pathogenesis, clinical picture, stages of development, tactics of treatment of NEC. Indications for surgical intervention, its nature, as well as complications are also reflected.

Necrotizing enterocolitis (NEC) of newborns (Necrotizing enterocolitis, necrotic enterocolitis, ulcerative necrotic enterocolitis, "disease of surviving premature infants) is a nonspecific inflammatory disease caused by infectious agents against the background of immaturity of local defense mechanisms and hypoxic-ischemic damage to the intestinal mucosa, prone to generalization of the process with the development of a systemic inflammatory reaction. The ICD code is P77. During the newborn period, NEC occurs on average in 4-6 children per 1000. About 82-93% of them are premature babies with low body weight. The disease usually begins on the 5-10 th day of a child's life, but in children with a body weight of less than 1000 g - after 3 months. The smaller the birth weight, the more often and later NEC can develop. B 1944 r. W. Heinrich (Switzerland) described 62 cases of necrotic enterocolitis in newborns; all children died. Only in 1969 J. Lloyd (8) in Detroit suggested that the development of the disease is associated with neonatal hypoxia. Currently, the mortality rate for this pathology ranges from 27 to 35%.

Etiology. NEC affects newborns who have suffered ischemic damage to the intestinal wall, intestinal dysbiosis, hyperosmolar nutrition. Predisposing factors are immaturity of a newborn with respiratory disorders, hypoxia, hypotension, hyperthermia, right-left shunt and congenital heart defects, or polycythemia, anemia. Umbilical vein catheterization, replacement blood transfusion, i.e. factors that cause low intestinal perfusion, have a certain effect. The combination of prematurity with immaturity of the immune response with impaired gastrointestinal function is important.

Gram-negative microorganisms (for example, intestinal and Pseudomonas aeruginosa, proteus, klebsiella, as well as anaerobic bacteria) are found in the peritoneal effusion of a child affected by NEC. In 22-30% of patients, microflora is seeded from the blood. No specific pathogen has been identified.

Pathogenesis. Spasm of mesenteric vessels, microembolism or thrombosis of small arterioles lead to local hypoxia and damage to the intestinal wall with subsequent violation of its integrity. Hypoperfusion dramatically reduces the resistance of the mucous membrane, its ischemic areas become more susceptible to the effects of microflora. However, intestinal wall

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ischemia is not the only pathogenetic factor in NEC. There is an increased permeability of the intestinal wall, anaerobes and (or) water-producing bacteria penetrate it and release gas (clostridium, bacteroids). These submucosal or subsurface accumulations of air form a pathognomonic symptom of necrotic enterocolitis in the form of intestinal pneumatosis (pneumatosis cystoides intestini).

Increased permeability facilitates the penetration of bacteria through the intestinal wall into the abdominal cavity and systemic blood flow. Most patients have all the signs of not only a local, but also a systemic inflammatory reaction, an increase in the concentration of cytokines in the blood.

Specific histopathological changes in the initial stages are edema and detachment of the epithelium of the intestinal villi. Then there are signs of destruction of the villi, swelling of the submucosa, after which the appearance of microhemorrhagia, microthrombs, blood stasis in the capillaries is characteristic. With a severe course of the disease, the complete disappearance of the structure of the villi, ulceration of the mucous membrane may occur; gas bubbles (pneumatosis) are visualized in the submucosa and subserosa. The serous membrane thickens due to edema.

Degenerative changes of the submucosal and intermuscular nerve plexuses are also detected. Plexuses lose glial cells and neurons. in the ganglia, lysis sites are revealed in the center, the ganglia resemble "empty baskets". Lesions resembling focal agangliosis are detected. The production of muscle tone inhibiting neurotransmitters (vasoactive intestinal peptide and nitric oxide) decreases. Changes in the submucosal plexus dominate, and they are more pronounced the more severe the damage to the mucous membrane.

Thus, degenerative changes of the intestinal nerve plexuses are observed in NEC. possibly secondary to hypoxemia.

With a decrease in blood flow to the tissues, the oxygen reserves necessary for adenosine triphosphatase (ATP) decrease, the cellular potential decreases, losing the ability to maintain an ionic gradient that provides transport through the cell membrane. There is a redistribution of calcium ions. Increasing its concentration activates the protease. converting xanthine dehydrogenase into xanthine oxidase. Depletion of cellular ATP reserves leads to an increase in the concentration of other adenine nucleotides. which are catebolized into adenosine, inosine, hypoxanthine and xanthine. These reactions lead to the "explosion" of superoxide, hydrogen peroxide and other toxic oxygen anionic radicals that can cause cellular destruction.

Another mechanism for reducing the production of superoxide is the suppression of xanthine oxidase by its inhibitor allopurinol. This agent blocks or reduces the conversion of hypoxanthine and xanthine into uric acid and superoxide. Not only intestinal cells are affected, but vascular elements, and, accordingly, the vascular wall.

With the progression of the process, transmural necrosis of the intestinal wall develops, followed by perforation. In some cases, multiple necrotic segments of the intestine are interspersed with affected, but viable areas.

In 50% of children, the small and colon are affected (most often the ileocecal region), in 25% - the colon, in 25% - the ileum. Rarely, the process captures the entire intestine.

Clinical picture. Early symptoms are often nonspecific: dehydration, a decrease in body temperature, a constantly swollen stomach, diarrhea often with blood, vomiting with an admixture of bile, apnea attacks, bradycardia. In parallel, there are gastrointestinal symptoms with constantly increasing bloating, fetid stool with an admixture of blood. After a time interval of varying duration, the patient's condition deteriorates sharply. Children become drowsy, the skin is pale gray, marbled and cold to the touch. The abdomen is tense, shiny, a pronounced vascular pattern is found. Phlegmon of the abdomen (edema and hyperemia), sepsis, metabolic acidosis, intravascular coagulation syndrome, respiratory failure soon follow, which indicate a far-reaching process of the disease.

In the dynamics of observation, the following stages of the disease are distinguished. The popular and often used classification of NEC by stages of flow, proposed by M. Bell et al. (1979) in modification by M. Walsh and R. Klieg-man (1986):

Stage 1 – "suspected necrotic enterocolitis" or initial manifestations:

1A - characterized by symptoms: unstable body temperature, apnea, bradycardia, lethargy of the child, slight bloating, hidden blood in the stool;

1B - along with the above, there is an admixture of blood in the feces.

Stage 2 - "obvious necrotic enterocolitis":

2A - "reversible stage", the appearance of signs of stage 1 + intestinal paresis + radiologically pneumatosis of the intestine;

2B - "irreversible stage", the same symptoms + radiologically gas in the portal system;

Stage 3 - "development of complications":

3A - signs of peritonitis, expansion of intestinal loops, multiple horizontal levels, pneumatosis, necrosis of the intestinal wall;

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3B - generalized peritonitis with its complications (disseminated intravascular coagulation syndrome, multiple organ failure syndrome, sepsis), pneumoperitoneum.

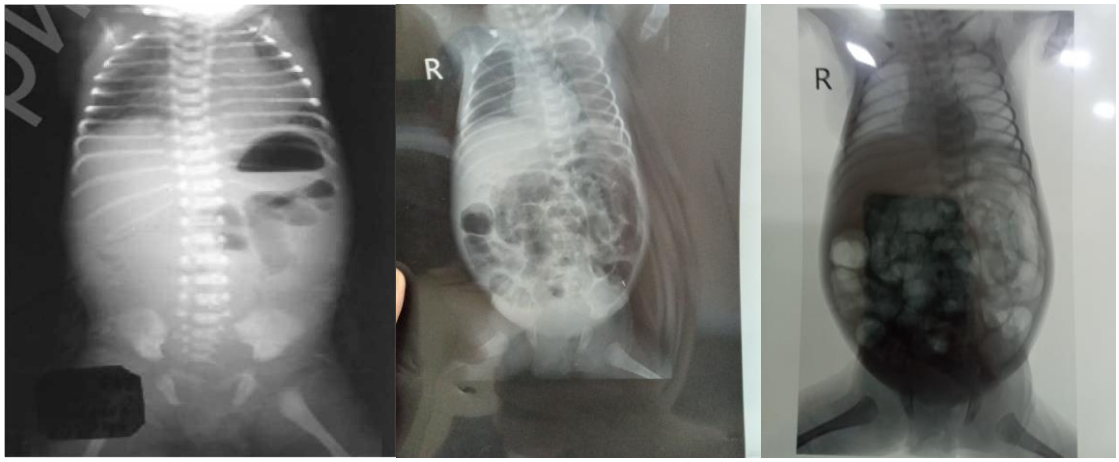
Diagnostics. Suspicion of the development of the disease involves a dynamic X-ray examination of the patient during the first day with an interval of 6-8 hours, then abdominal radiographs are performed according to indications. Radiological signs of NEC: uneven swelling of intestinal loops, pneumatosis of the intestinal wall (cystic or linear), gas in the portal system of the liver, the presence of a hypostatic loop of the intestine, pneumoperitoneum, the presence of fluid in the abdominal cavity. Intestinal pneumatosis and gas in the portal system of the liver.

Radiological characteristics of NEC at each stage of the disease according to M. Bell (1979) and M. Walsh and R. Kliegman (1986):

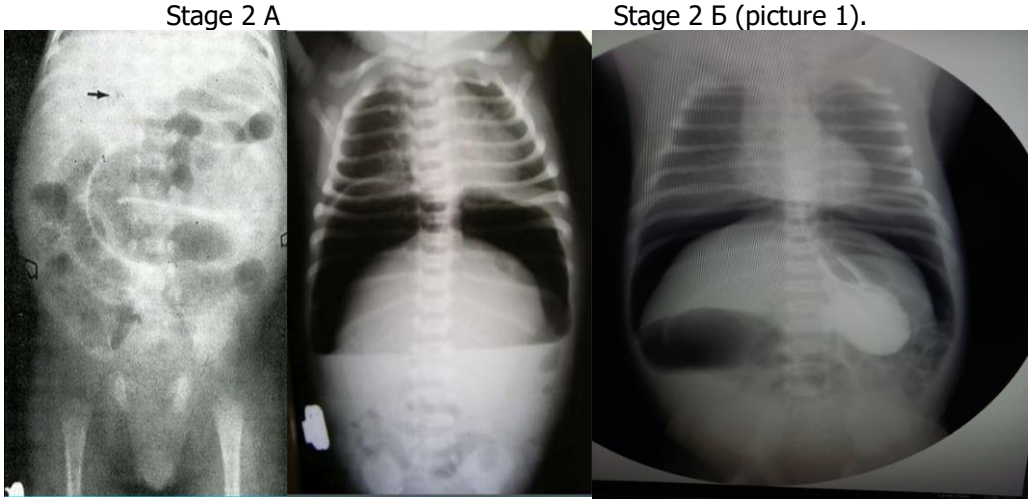
Stage 1A: dilation of intestinal loops is the most common symptom, it was already detected in 65% of newborns in the early stage of the disease. A significant expansion of intestinal loops and the appearance of horizontal levels indicates the severity of the disease and its progressive course.

Stage 1B: the presence of a fixed (static) bowel loop that does not change its position on repeated images (straightened, rigid bowel loops). This sign indicates intestinal ischemia and occurs in 15% of cases. Such loops can be localized, single, or multiple. Comparison of radiographs taken at a certain time interval (after 6-8 hours) helps to differentiate the usual expansion of intestinal loops with fixed and fixed sites (Fig.3)

Stage 2A: gastric dilatation is a symptom of NEC due to both gastric atony caused by exposure to bacterial toxins and pseudo-obstruction of the pyloric department as a result of mucosal edema occurs in 7-10% of children with NEC.



Stage 2 A Stage 2 Б (picture 1).



Stage 3 A (picture 2)

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Stage 2B: decreased pneumatization and uneven gas filling of intestinal loops is observed in 30-40% of patients. This symptom is explained by the fact that as a result of active peristalsis, intestinal contents move from viable parts of the intestine to ischemically affected, in which peristalsis is either reduced or completely absent. Hypopneumatization of the intestine is shown in Figure 5.

Stage 3A: The sudden appearance on radiographs of signs of ascites detected in 11% of patients is an unfavorable factor indicating the reaction of the peritoneum to bacterial peritonitis, a possible covered perforation of the intestine.

Stage 3B: Pneumatosis of the intestinal wall (intramural accumulation of gas). This symptom is characteristic of stage 3 of the disease. Pneumatosis is determined approximately in 18% of cases. Pneumatosis does not always precede intestinal perforation. 90% of patients have pneumatosis of the intestinal wall. Pneumatization of the intestinal wall is a reliable sign of NEC and is recognized during surgery against the background of swollen intestinal loops and during X-ray examination. Gas in the portal vein is a reliable sign indicating a far-reaching process that predetermines an unfavorable prognosis. This sign occurs in 3% of cases. It is assumed that the gas in the form of emboli enters the portal vein system from the necrotic intestinal wall (with pneumatosis) or is formed directly in the vascular capillaries as a result of the penetration of gas-producing bacteria (anaerobes) into these capillaries.

The presence of gas in the portal vein system occurs in 61% of children with total intestinal necrosis and determines an unfavorable prognosis, therefore the presence of this symptom is an absolute indication for surgical treatment.

Pneumoperitoneum - accumulation of free gas in the abdominal cavity, more often under the domes of the diaphragm, irrefutably indicates perforation of the hollow organ and is characteristic only for stage 3B of the disease and is detected in 98-99% of newborns with NEC. The absence of free gas in the abdominal cavity on the radiograph can be observed in the case of "covered" perforation, atypical localization of the hole in the bag. Pneumoperitoneum can occur without perforation of the hollow organ in weakened patients, many of whom were on artificial ventilation for pulmonary pathology (barotrauma leads to rupture of the alveoli and penetration of air through the mediastinum into the abdominal cavity). This situation occurs in 10-15% of cases. This point should be taken into account in the differential diagnosis of necrotic enterocolitis. Recently, much attention has been paid to increasing intra-abdominal pressure in progressive necrotic enterocolitis and the development of intraabdominal hypertension syndrome. In 2004, at the WSACS (World Society of the Abdominal Compartment Syndrome) conference, intra-abdominal hypertension was defined as a steady increase in intra-abdominal pressure up to 12 mmHg or more, which is recorded at least with three standard measurements with an interval of 4-6 hours. Intraabdominal hypertension is a prodromal phase of the development of abdominal inflammatory syndrome. Intraabdominal hypertension syndrome is a multiple organ failure caused by an increase in intra-abdominal pressure in a confined space, which leads to circulatory disorders, hypoxia and ischemia of organs and tissues located in this space, contributing to a marked decrease in their functional activity until its complete cessation. An informative method for diagnosis is sonography, which allows to identify the expansion of intestinal loops, delamination and thickening of the intestinal wall, free fluid and gas in the abdominal cavity. Ultrasound examination in Doppler mode allows to assess the blood flow in the vessels of the upper mesenteric artery basin and in some cases to carry out a clear gradation of the stages of necrotic enterocolitis, the transition to necrosis of the intestinal wall, which requires active surgical tactics.

Among diagnostic laboratory tests, the most constant are changes in the hemogram (leukocytosis or leukopenia, a shift of the leukocyte formula to the left, thrombocytopenia), an increase in the content of C-reactive protein, acidosis, electrolyte imbalance. Unfortunately, all of them are not specific to this pathology.

According to foreign authors, the role of inflammatory markers in the development of necrotizing enterocolitis is being actively studied. Among a large group of potential molecular prognostic factors, a special place is occupied by the system of matrix metalloproteinases, lactoferrin, calprotectin, protein binding fatty acids.

If there is a sign of free fluid in the abdominal cavity and perforated peritonitis, surgical intervention is indicated, since with timely surgery, the chances of recovery increase significantly. The operation consists of a transverse laparotomy, lavage of the abdominal cavity, resection of the necrotic part of the intestine and the imposition of one or more enterostomes. Resections should be carried out sparingly so as not to create a situation with a short intestine.

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CONCLUSIONS

1. NICK affects most often newborns with a low body weight from 500 to 1550 g. and 32 weeks of gestation, who have suffered ischemic damage to the intestinal wall, dysbiosis, hypeosmolar nutrition. At the same time, there is an increased permeability of the intestinal wall, anaerobes penetrate into it and release gas (intestinal pneumatosis), then colonize pathogenic bacteria, causing enteritis and peritonitis.

2. During the course of the disease, there are 3 stages: stage "suspected necrotic enterocolitis" or initial manifestations, stage II "obvious necrotic enterocolitis", stage III "development of complications". The clinical and radiological picture of each stage are different and require appropriate treatment tactics from the doctor.

3. Surgical treatment is indicated in stage III. It boils down to a transverse laparotomy, sanitation of the abdominal cavity, resection of the affected intestine, the imposition of enterostomy. Restoration of intestinal patency is planned in 6-8 weeks.

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